Listing of Claims:

The following claims are currently pending in this application.

1. (**Previously presented**) A method for proliferating cardiomyocytes comprising a step of introducing
(a) cyclin,
(b) cyclin-dependent kinase, and
(c) one or a plurality of a gene encoding a factor that inhibits the production, function or action of Cip/Kip family protein, into cardiomyocytes *in vitro*, and a step of subsequently culturing or maintaining said cells.

2. (**Canceled**)

3. (**Withdrawn**) A method for proliferating cardiomyocytes comprising a step of introducing
(a) cyclin,
(b) cyclin-dependent kinase, and
(c) one or a plurality of a gene encoding a factor that inhibits the production, function or action of Cip/Kip family protein, or a nucleic acid that inhibits the production of Cip/Kip family protein, into cardiomyocytes *in vivo*, and
a step of subsequently maintaining said cells.

4. (**Previously Presented**) The method of claim 1, wherein said cyclin is a cyclin that activates CDK4 or CDK6 of mammals.

5. (**Original**) The method of claim 4, wherein said cyclin is cyclin D of mammals.

6. (**Previously Presented**) The method of claim 1, wherein said cyclin-dependent kinase is a cyclin-dependent kinase to be activated by cyclin D.

7. (**Previously Presented**) The method of claim 6, wherein said cyclin dependent kinase is CDK4 or CDK6.

8. (**Previously Presented**) The method of claim 1, wherein the Cip/Kip family protein is $p27^{Kip1}$.

9. (**Previously Presented**) The method of claim 1, wherein the factor that inhibits the production, function, or action of Cip/Kip family protein is a factor with an action to promotes the degradation of the Cip/Kip family protein.

10. (**Original**) The method of claim 9, wherein the factor with an action to promote the degradation of the Cip/Kip family protein is a component of ubiquitin ligase.

11. (**Previously Presented**) The method of claim 10, wherein the component of ubiquitin ligase is an F-box factor that binds to the Cip/Kip family protein.

12. (**Original**) The method of claim 11, wherein the F-box factor capable of binding to the Cip/Kip family protein is Skp2.

13. (**Withdrawn**) The method of claim 1, wherein the nucleic acid that inhibits the production of Cip/Kip family protein is siRNA specific to a gene encoding the Cip/Kip family protein.

14. (**Withdrawn**) The method of claim 13, wherein the nucleic acid that inhibits the production of Cip/Kip family protein is siRNA specific to the $p27^{Kip1}$ gene.

15. (**Previously Presented**) The method of claim 1, comprising introducing the genes into cardiomyocytes, using a viral vector or liposome.

16. (**Previously Presented**) The method of claim 1, wherein at least one of the cyclin gene and cyclin-dependent kinase gene is tagged with a nucleotide sequence encoding a nuclear localization signal.

17. (**Previously Presented**) A vector comprising
    (a) a cyclin gene
    (b) a cyclin-dependent kinase gene, and
    (c) one or a plurality of a gene encoding a factor that inhibits the production, function, or action of Cip/Kip family protein.

18. (**Previously Presented**) The vector of claim 17, wherein the cyclin is a cyclin that activates CDK4 or CDK6 of mammals.

19. (**Original**) The vector of claim 18, wherein the cyclin is cyclin D of mammals.

20. (**Previously Presented**) The vector of claim 17, wherein the cyclin-dependent kinase is a cyclin-dependent kinase to be activated by cyclin D.

21. (**Original**) The vector of claim 20, wherein the cyclin-dependent kinase is CDK4 or CDK6.

22. (**Previously Presented**) The vector of claim 17, wherein the factor that inhibits the production, function, or action of Cip/Kip family protein is a factor with an action to promote the degradation of the Cip/Kip family protein.

23. (**Original**) The vector of claim 22, wherein the factor with an action to promote the degradation of the Cip/Kip family protein is a component of ubiquitin ligase.

24. (**Original**) The vector of claim 23, wherein the component of ubiquitin ligase is an F-box factor capable of binding to the Cip/Kip family protein.

25. (**Original**) The vector of claim 24, wherein the F-box factor capable of binding to the Cip/Kip family protein is Skp2.

26. (**Withdrawn**) The vector of claim 17, wherein the nucleic acid that inhibits the production of Cip/Kip family protein is siRNA specific to a gene encoding the Cip/Kip family protein.

27. (**Withdrawn**) The vector of claim 26, wherein the nucleic acid that inhibits the production of Cip/Kip family protein is siRNA that is specific to $p27^{Kip1}$ gene.

28. (**Withdrawn**) The vector of claim 17, wherein at least one of the cyclin gene and cyclin-dependent kinase gene is tagged with a nucleotide sequence encoding a nuclear localization signal.

29. (**Withdrawn**) A pharmaceutical composition for use in a treatment of cardiac disorder comprising the vector of claim 17.

30. (**Withdrawn**) The pharmaceutical composition of claim 29, wherein the cardiac disorder is myocardial infarction, ischemic heart disease, congestive heart failure, hypertrophic cardiomyopathy, dilated cardiomyopathy, myocarditis, or chronic heart failure.

31. (**Previously Presented**) Cardiomyocyte obtained by the method of claim 1.

Claims 32-33 (**Canceled**)

34. (**Previously Presented**) A method for proliferating cardiomyocytes that have withdrawn from the cell cycle comprising a step of introducing

    (a) cyclin,

    (b) cyclin-dependent kinase, and

    (c) one or a plurality of a gene encoding a factor that inhibits the production, function or action of Cip/Kip family protein, into cardiomyocytes *in vitro*, wherein said cardiomyocytes have withdrawn from the cell cycle, and a step of subsequently culturing or maintaining said cells.

35. (**Previously Presented**) The method of claim 34, wherein the cardiomyocytes are adult cardiomyocytes.